CLINICAL LECTURE

ON A

CASE OF CHRONIC CEREBRAL MENINGITIS: CHRONIC ABSCESS OF BRAIN: LIMITED PERITONITIS: OBSTRUCTION OF THE PORTAL AND SPLENIC VEINS: AND ACUTE CEREBRO-SPINAL MENINGITIS.

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THE subject was M. M., aged 37, of no occupation. The father of the patient, who was otherwise a healthy man, was said to have had several attacks of temporary paralysis affecting the right leg and both arms. These seizures came on suddenly, and their effects lasted from a few days to several months. Otherwise, the family history was healthy. The patient himself, a gentleman of large property, appeared to have enjoyed perfectly robust general health all his life till the present illness. He was born in Australia, and remained there till a few years ago, engaged in active and healthy pursuits. Since his residence in England, he had passed his time in country and outdoor occupations, and had always been noted for his activity and strength, as well as for intellectual acumen. At the age of fifteen, on awaking one morning, the patient discovered weakness in one of his arms; this soon passed away. Since then, he had been liable to attacks of a similar nature. Being in his usual good health at night, next morning he would find one or more of his limbs on the left side weak. This paresis lasted from a few days to some weeks, and on one occasion as long as six months. The paralysis was not complete; and, when it affected the leg, did not prevent the patient from walking, but only caused him to be lame and drag the toes along the ground. When the arm was attacked, it was weak; and there was numbness, especially on the ulnar side. In the intervals between these seizures, he was perfectly well, and there was no trace of impaired motion. Even when the paresis was present, he could, as a rule, take a great amount of exercise; but on several occasions, when it was severe, he was completely laid up. He had never had syphilis or or any other cerebral symptoms, and was not aware of having had "fits" or attacks of any kind.

About the middle of September 1880, the patient, being in his usual good health, was seized with a severe pain in the epigastric region, and with an illness which laid him up in bed for six weeks. This pain was extremely acute and continuous, so that he had to be kept more or less under the influence of opium. It appeared to be confined to a spot about the ensiform cartilage, was increased on pressure and after food, and was modified by position. There was a little vomiting; but no blood was ever seen in the ejected matter. The medical men in attendance believed the affection to be ulcer of the stomach. At the end of six weeks, the symptoms passed away; the patient become convalescent; travelled to London; and for the next three weeks, although generally weak, considered he was rapidly improving in every way.

On November 21st, the patient was seized with a severe pain in the epigastrium, which again compelled him to apply for medical assistance.

Present Condition.—The patient was very tall (said to be 6 feet 5 inches in height), of spare yet robust build, pale and somewhat thin from his recent illness. He complained of a severe dull aching pain at the ensiform cartilage. This was not influenced by pressure, but was increased after food, although not to a great extent. There was nothing to be seen or felt locally. There was no vomiting, nor other symptoms. All the organs were healthy; but the patient was slightly feverish, greatly depressed in spirits, and had a look of much anxiety and suffering. This condition continued without change during the next five days, the pain occurring in paroxysms of an extremely severe character, leaving during the intervals a dull aching sensation. On November 25th, the urine was found loaded with bile; and on the following day the patient presented all the usual appearances of jaundice. For the following three weeks, the condition remained much the same; and there was almost constant pain in the epigastrium, increased frequently by paroxysms of a very acute character. There were also griping pains in the abdomen, especially before and after a stool. The bowels were constipated, and there was well-marked jaundice; no vomiting or headache; no œdema or anasarca; and the appetite was fairly good. The most careful and repeated physical examinations failed to detect anything abnormal. About December 19th, all the symptoms gradually began to diminish in severity; abundant bile appeared in the stools; the urine became clear; the yellow colour of the skin perceptibly faded; and the epigastric pain was much relieved. This general improvement so advanced, that in a few days the patient became quite convalescent, was able to sit up, and, with the exception of great weakness and emaciation, felt in good health and spirits. This continued till January 1st, when he was suddenly seized with an acute lancinating pain in his head, accompanied with feverishness and great depression, which lasted during the day, and which was finally relieved by a hypodermic injection of morphia. A few days subsequent to this, a return of his former complaint was observed; namely, paresis of the left leg. The extensors of the foot were paralysed; and, when the patient walked, he was very lame, and the toes dragged along the ground. The extensors of the thigh were also weak. All the flexor muscles seemed to be normal. The extension of the left hand was somewhat impaired, but otherwise the limb was unaffected. There was no loss of sensibility. With this exception, the general convalescence progressed favourably till January 6th, when the patient stated that he had a "fit". He said he had a repetition of this seizure on the following day. During the succeeding twenty-four hours, he had two more attacks, one of which was witnessed by Mr. W. L. Purves (who watched the case with me throughout), and who described it as a genuine epileptiform fit. There were sudden convulsions of all the limbs and face, and complete loss of consciousness, lasting for some minutes. Immediately afterwards, the patient was weak and depressed, otherwise well. Next day, there was distinct paresis of the right arm, and the face was drawn slightly to the left; no other cerebral symptoms. This paresis of the arm gradually increased till January 12th, when its movements were very feeble and imperfect, and the grasp of the hand was almost lost. The patient then was depressed and emotional, but his intelligence was perfect. There were now slight thickness of speech and hesitation in articulating. On the following day, the right arm was absolutely paralysed as to motion, but the sensibility was unaffected. Two days later, the right leg was found weak; there was considerable difficulty in articulation, and the patient spoke in a mumbling way. The movements of the tongue were sluggish; and, when protruded, it was pushed towards the left side. The face was distinctly drawn towards the left. In addition to the difficulty of articulation, there was evidence of partial true aphasia, as the patient had difficulty in selecting and recollecting suitable words with which to express himself; but he never employed wrong ones. His understanding seemed intact. Till January 17th, all these symptoms rapidly increased, when, in addition, he was attacked with severe pain in the back, with cramps and contractions of the legs. The patient was dull and apathetic, but appeared to understand what went on around him. His speech was now unintelligible. All the symptoms increased, and on January 22nd the whole of the right side was completely paralysed. Although the patient could not say a word, he appeared intelligent, and understood what was said to him. On the 28th, the patient was evidently becoming weaker, the sphincters began to be relaxed, and the catheter had to be used. From this date to February 8th, he gradually sank, became comatose, and died.

POST MORTEM EXAMINATION.—This was made by Dr. Goodhart, in my unavoidable absence; and the following is an abstract from his excellent report. *Head.* The cranial bones, dura mater, and sinuses were normal. The arachnoid and pia mater were thickened, and at the base much so; they were opaque, and their vessels were imbedded in thick white fibrous tissue. This induration of the membranes was symmetrical, except at the upper part of the right ascending parietal convolution, immediately adjacent to the longitudinal fissure, where there was a small patch of scar-like fibrous tissue in the pia mater, below which the cortical substance appeared healthy. The brain was of normal size; the surface was dry and greasy-looking, apparently the result of acute meningitis. The Sylvian fissures were adherent. The convolutions upon the right side looked normal; but on the left they were much flattened, and the sulci were indistinctly marked. On the surface of the left ascending frontal convolution, bordering upon the longitudinal fissure, appeared a rounded eminence, about half an inch in diameter, and of yellow colour, to which the dura mater and arachnoid membranes were adherent. A section parallel to the longitudinal fissure was made through this swelling, when it was found to communicate with an abscess below containing from an ounce and a half, to two ounces of thick, green, tenaceous, odourless pus. The cavity of the abscess was lobulated, and about the size of a small hen's egg. It occupied the white substance of the brain, immediately below the ascending

frontal and parietal and the posterior portions of the frontal convolutions. Above, it appeared to point at the eminence already described at the upper part of the ascending frontal convolutions; and below it reached very near the convolutions of the island of Reil. The interior of the cyst was smooth, and covered with flaky pus. That portion of the abscess which approached the cortex had no distinct cyst-wall; but the remainder was surrounded by a dense tough capsule about a millimètre in thickness. The outer layer of this appeared very vascular, and in some parts of it there were small extravasations of blood. This capsule separated readily from the surrounding brain-tissue, and could easily have been enucleated. The ventricles and ganglia were healthy, except that the latter were somewhat compressed on the left side. Otherwise, the brain was normal. *Spinal Cord.* A quantity of turbid fluid was found beneath the sheath. There was general injection of the vessels of the cord, and a quantity of yellow lymph was found in the meshes of the pia mater. The cord was soft, and the white matter streaky-looking from injection of the vessels. *Abdomen.* Above the umbilicus, the omentum was adherent to the abdominal wall. There were a number of tough fibrous bands of adhesions between the stomach and under surface of the liver. All the structures of the portal fissure were imbedded in a mass of dense fibrous tissue, making it a difficult task to dissect out the various channels. When this was effected, the portal vein was found to be completely obliterated by a firm organised fibrous thrombus, strongly adherent to the vein-wall. The splenic and mesenteric veins at their upper part were obstructed in a similar manner. The common bile-duct permitted bile to flow through it readily; and its walls were rugose-looking and dilated, but otherwise healthy. The gall-bladder was healthy, and no trace of stone was found in its interior. At the lower omentum was a regular plexus of minute veins, which made their way to the cardiac end of the stomach, and discharged themselves into enlarged œsophageal veins. Some also appeared to run backwards from the portal vein and communicate with the inferior vena cava. Another large plexus of veins passed from the sigmoid flexure to the internal abdominal ring, and blended with the spermatic vein, which was unusually large. The *Liver* was in structure normal. The branches of the hepatic artery were enlarged and thickened, and the branches of the portal vein were not discoverable to the naked eye. The *Stomach* was considerably mammillated, otherwise normal. The *Spleen* weighed six ounces, and was somewhat tough and bloodless. The artery was normal, but the vein could not be discovered. *Lungs.* There was some recent lymph in the lower portions of the pleural cavities on both sides. Both lungs were extensively affected with bronchopneumonia. The other organs and structures of the body were normal.

COMMENTARY.—This case presents many features of pathological and clinical interest; but I shall limit my remarks to the following three general heads: 1. The lesion of the portal system; 2. The lesions of the nervous system; and 3. The relation existing between these two morbid states.

1. *The Lesion of the Portal System.*—Thrombosis of the portal vein, although by no means common, is a clearly recognised pathological condition, and a considerable number of cases have been recorded both at home and abroad. In the instance before us, this state was probably the result of a limited peritonitis, involving the structures lying in the portal fissure. After death, the tissues in this neighbourhood were found thickened and matted together: hence, either by mechanical pressure or by inflammatory irritation of the venous wall, or possibly by both these causes acting together, the blood in the interior of the vessel during life had coagulated, and the clot had subsequently become organised, so as to form a complete obstruction to the vein. As a result of this mechanical condition, certain symptoms are usually induced—namely: ascites, enlargement of the spleen, dilatation of the veins, diarrhœa, hæmorrhage, œdema, etc.; but, in this case, all these symptoms were conspicuous by their absence. Here the only evidence of any abdominal affection was intense paroxysmal pain in the epigastrium, and jaundice, which for a time gave us the impression that the patient was suffering from gall-stones. There were absolutely no symptoms to point to any obstruction of the portal vein. This serves to show that, when this condition is induced gradually, the circulation must be established in other directions, so that the usual mechanical effects are prevented. In the healthy subject, the anatomist finds that there are minute veins which pass directly from the intestines into the liver, independently of the ordinary portal system. These are accessory portal veins. There are also numerous venous communications between the portal system and the inferior and superior venæ cavæ; and, in short, even in health, there is a free anastomosis of all the vessels in this neighbourhood. When the main trunks of the portal vein are obstructed, these collateral branches enlarge, and thus establish the circulation. In the present case, this was actually discovered; and, as far as a dissection of the matted tissues could be made, a number of these enlarged collateral branches were demonstrated. We may therefore assume, that it was owing to the successful re-establishment of the circulation that the absence of the usual mechanical symptoms was due. The jaundice during life was probably the result of pressure on the gall-duct by the inflamed and thickened peritoneum.

2. *The Lesions of the Nervous System.*—The patient, from the age of fifteen, had been constantly seized with attacks of temporary paralysis, usually affecting the left side of his body. He generally discovered these symptoms on awaking in the morning; and the paresis, in different attacks, lasted from a few days to several months—the patient during the intervals being in a perfectly normal condition. The movements of the limbs were not abolished, but were sufficiently impaired to cause great weakness of the left arm and hand, and dragging of the leg, and certain muscles seemed to be more affected than others. The patient was ignorant of having had any kind of fit. The nature of these seizures is not clear; but, from the fact that his father suffered from exactly similar symptoms, it might be urged that they may have been the results of epileptic attacks occurring during sleep, and therefore without the knowledge of the patient. On the other hand, after death, there was found old-standing meningitis, and more especially a patch of inflammatory thickening of the membrane over the right ascending parietal convolution. It is possible that these pathological conditions may have existed for many years, and given rise to occasional attacks of paralysis on the left side. Until five weeks before death, with the above exceptions, there had been no other cerebral symptoms. At this time, the patient was suddenly seized with violent pain in the head and feverishness, followed a few days later by epileptic attacks, subsequently by paralysis of the right side, including the face and tongue; followed by true aphasia; and, finally, by coma and death. On examination, an abscess was found in the left side of the brain, whose size and position was sufficient to satisfactorily account for the paralytic and functional symptoms on the right side of the body. These last, however, made their first appearance only five weeks before death; and, on inspecting the abscess, the question arises as to whether its age corresponds with the symptoms observed during life. To judge the length of time an abscess has existed, is often extremely difficult. When acute or subacute, there is no capsule surrounding it; therefore, when such exists, as in the present case, we may assume it to be chronic. Meyer states that the earliest appearance of a capsule is at the sixth or seventh week; and Huguenin demonstrated the distinct evidence of one not earlier than the twelfth week. In the patient under notice, the capsule was thick, dense, and well defined, and otherwise the abscess presented all the appearances of a chronic nature, and therefore must have existed at least for several months. As the earliest symptoms of any brain disturbance were noticed for the first time only five weeks before death, we must assume that the abscess existed prior to that period, without producing any objective phenomena. The temporary attacks of paralysis, from which the patient suffered all his life, had apparently no connection with the cerebral abscess, as they affected the left side of the body; whereas the abscess was on the left side of the brain, and subsequently induced right hemiplegia. We are therefore left in doubt as to the date of the origin of the abscess. We can only assume that it must have existed at least some months, before there were any trace of symptoms resulting from it. Such cases are very rare: for, although it is not uncommon to find cerebral abscess having existed, apparently even involving a motor area, without producing sufficiently definite symptoms to permit a diagnosis being arrived at, still there are almost always some phenomena present pointing to impairment or alteration of brain function. In the present case, the most careful investigation of the patient and his friends failed to elicit the existence of any cerebral symptoms whatever, prior to the terminal changes occurring shortly before death. These final symptoms presented nothing remarkable, consisting as they did of impaired function of the left side of the brain, evidently due to recent extension of the cavity of the abscess causing pressure and altered circulation in the hemisphere. This was demonstrated by the morbid appearances seen after death, as already described. Upon the question of cerebral localisation, this case throws little light. Supposing the abscess to have existed for a certain period without producing symptoms, it only indicates that a slowly growing lesion, occupying the position of motor conducting fibres, need not of necessity cause paralysis. There is no evidence that, during the latent stage, any portion of the cortical matter was involved—nor did it appear that any of the important ganglia were directly affected. The abscess was situated in the medullary portion of the brain, immediately below what we have recently been told are the motor centres; and, although it might be assumed that the nervous communications would thus be severed, yet, in this instance, no paralysis ensued. Later, we have pathological demonstration of extension of the cyst-wall, of softening of

the grey matter, and pressure on the convolutions and deep-seated organs, which conditions were accompanied by paralysis and other signs of functional inactivity. A small patch of cicatricial tissue was found over the upper part of the right ascending parietal convolution, close to the longitudinal fissure; and we know that all his life the patient was liable to altered motility of the left side, and especially of the leg. If it could be proved that these were cause and effect, this would support recent observations by Ferrier and others on cortical localisation.

3. *The Relation between the Abdominal and Cerebral Lesions.*—Two comparatively rare diseases—namely, obstruction of the portal vein, and abscess of the brain—having occurred in the same individual, the question arises: Was each of these lesions independent of one another? or, were they in any way related? Cases are on record in which abscess of the brain seemed to have originated as a result of abdominal disease; but these are very rare, and were usually of a pyæmic nature. We have seen that the first symptoms of abdominal disease began about five months before death. Although there is no proof of it, it is possible that the abscess originated at this time. If so, could the limited peritonitis and obstructed portal system have been its cause? Of seventy-six cases of abscess of the brain, collected by Sir William Gull and Dr. Sutton, in only three could the disease be traced to an abdominal affection; and in each of these there was a collection of pus in that cavity. Suppuration in any part of the body may occasionally lead to abscess of the brain, more especially if pyæmic in character. In the case before us, there was no evidence of any suppuration having taken place, either in the abdominal cavity, or in any other part of the body; and there never had been any appearance of blood-poisoning or other constitutional disturbance, sufficient to account for the existence of a cerebral abscess. It has been suggested that this lesion was of an embolic origin, due to the abnormal condition of the portal circulation. We can, however, scarcely imagine an embolus, filtering through the liver and lungs, capable of producing mechanical obstruction; and, if the particles were sufficiently minute to effect a passage, we have no reason to suppose they should create mischief specially in the brain, unless they were poisoned, which the facts of the case disprove. In this instance, the cause of the abscess is not apparent, and therefore its age is uncertain. There was no injury, disease of the ear, or symptoms of an acute cerebral attack, which are the most common causes. Of the seventy-six cases already cited, the abscess could be traced to disease of the ear in twenty-seven; to injuries of the head in seventeen; to lung-disease in ten; to surgical affections in nine; and in eight only was no definite cause ascertained. To such so-called idiopathic abscesses, we may add this one, as the data before us fail to establish either its cause or age. We, therefore, fail to prove any relation between the abdominal and cerebral symptoms.

The facts of this case, taken as a whole, seem to suggest the following sequence of events. The patient, in early life, without apparent cause, was afflicted with chronic cerebral meningitis, which, for many years, caused no symptoms except occasional attacks of temporary hemiplegia. The changes in the tissues and circulation, thus induced, may have afterwards been the starting-point of the chronic abscess, which existed for a long time without producing any symptoms. Five months before death, the patient was seized with an acute attack of limited peritonitis, involving the portal system, and subsequently causing thrombosis and obstruction of these veins. The constitutional disturbances, induced by the second attack of this illness, probably excited the recent acute action in the brain, in the shape of cerebro-spinal meningitis, and the extension of the already existing abscess.

FUNGOSITIES OF THE FEMALE BLADDER.—Dr. Atlee publishes, in the *Boston Medical and Surgical Journal* of March 30th, a case which is specially interesting at the present time, as Sir Henry Thompson has so recently, at the Royal Medical and Chirurgical Society, drawn attention to tumours of the bladder. Dr. Atlee saw his patient in September 1880; she was a lady, aged nineteen; she was obliged to pass urine every half hour, and the urine contained a large quantity of blood. An exploration of the bladder was at once advised, and submitted to, under the influence of anæsthetics. An ordinary pair of dressing-forceps was introduced into the urethra, opened, and withdrawn; this was done several times, and the urethra thereby fully dilated. On the introduction of the finger into the bladder, no calculus or distinct tumour was to be felt; but "about the fundus were a number of fungosities, or soft growths, some of them more than a half inch in length, and about one line in thickness." Dr. Atlee scraped them away with his finger-nail, and up to the date of the paper (March 1st, 1882) the young lady has remained perfectly cured. Dr. Atlee is particular to explain that the growths were not villous, but true fungosities, having anatomically the same fundamental structure as the mucous membrane from which they sprang.

REMARKS ON ACUTE SPINAL PARALYSIS.

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WE must first ask what we mean by "acute spinal paralysis". The term has become restricted to a narrower range of symptoms than it literally designates. Most acute lesions of the cord produce "acute spinal paralysis", but by the expression is commonly understood only those forms in which the paralysis is local in its distribution, and is accompanied by rapid muscular wasting. It thus includes only the disease with which we are familiar as "infantile paralysis", and the analogous affection which occurs in adults. Pathological observation has demonstrated that, in most cases, the symptoms are due to an acute inflammation of the anterior grey cornua of the cord; hence the term suggested by Kussmaul, of "anterior poliomyelitis". We may also, if we wish for a descriptive pathological name of still simpler character, call it "cornual myelitis". But it is important to bear in mind that these are pathological, while "acute spinal paralysis" is a clinical name, and they are not quite conterminous. The symptoms of acute spinal paralysis may depend on any acute process in the anterior cornua. They may occur, for instance, from hæmorrhage in this situation as well as from inflammation. We must, therefore, include under the term acute spinal paralysis, cases which are not myelitis.

The general symptoms of acute spinal paralysis are well known, since, as it affects children, the disease is familiar to all practitioners; and its characters in adults are nearly the same. There is an acute, or subacute, onset, with symptoms of a general illness. The paralysis is usually at first extensive, sometimes universal; but power gradually returns, except in a limited region, in which the muscles rapidly waste, and present the electrical reactions which characterise nerve-degeneration—loss of irritability to faradisation; preservation, and even increase, of irritability to voltaic electricity, with certain alterations in the mode of response to the latter.

Our knowledge of the nature of the process in the spinal cord is, perhaps, more meagre than in the case of any disease equally common. We know nothing of the way in which, in ordinary cases, the inflammatory lesion commences. Death during the early stage of the affection is extremely rare, and opportunities for examination have been so scanty, that there exists no observation on the state of the spinal cord during the first few days of the affection. It is possible, however, that more cases in children are fatal at the onset than is commonly believed, because the nature of the disease at this period may readily be mistaken. It is chiefly to general practitioners that we must look for the opportunity of further investigation on this important point.

It is singular that our knowledge of the causes of the disease is hardly more definite than that of its pathology. Even the causes which are recognised are not altogether beyond question. Hereditary tendency to nervous affections seems to have but little influence in its causation; most authorities doubt the influence altogether. I have myself been strongly impressed by two or three cases, in which other members of the families have suffered from other acute affections of the nervous system. This is a point on which a comparison of experience is very desirable.

The influence of age is well known, but the remarkable proclivity of children to suffer from the disease is quite unexplained. They do not suffer specially from other inflammatory affections of the nerve-tissues, although liable to certain inflammations of the membranes. The disease has been observed to come on a few days after birth. If the account given by the friends of one of my patients be correct, the disease came on, in her case, *in utero*. On the other hand, recent medical literature abounds with examples occurring in adult life. I have seen one case, in which the disease came on in a man of more than seventy years of age. After some rheumatoid pains about the neck and shoulder, he found the right hand weak; and certain muscles, especially the long extensors of the finger and thumb, and, to a less extent, the thenar and hypothenar muscles, rapidly wasted, and presented the characteristic electrical reactions. The hand also presented a well marked condition of myxœdema. I am not aware that this condition has been before observed in connection with the disease.

In young children, it is customary to refer the disease, like many others, to the irritation of teething. Is there the slightest ground for this? Here, again, we want more facts, which only the general practitioner can supply. We want to know the age and the state of dentition at the time of the onset. In a large number of cases, this information can only be given by those who have the charge of the patients